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Changes in electromyographic activity of trunk muscles within the sub-acute phase for individuals deemed recovered from a low back injury.

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Abstract

Evidence indicates that previous low back injury (LBI) is a strong predictor for re-injury. The purpose of this study was to examine whether neuromuscular patterns remain altered in a LBI group who were deemed recovered. Surface electromyograms from 12-abdominal and 12-back extensors sites and motion variables were recorded from 33 LBI individuals (sub-acute phase) and 54 asymptomatic controls. Pain-related variables were recorded and a clinical assessment performed for LBI participants. Subjects performed a symmetrical lift and replace task in two reaches. Pattern recognition techniques were applied to normalized activation amplitude patterns to extract key recruitment strategies. Mixed model ANOVAs tested for effects ($p < 0.05$). Despite similar task performance, significantly ($p < 0.05$) different recruitment strategies were observed for the LBI group. There were higher activation amplitudes for LBI subjects in all muscles (except posterior external oblique) and greater co-activation between abdominal and back extensor sites compared to controls. Local abdominal and back extensor sites showed altered responses to increased physical demands in the LBI group. Despite outcomes indicating recovery, the LBI group had altered neuromuscular patterns compared to asymptomatic controls supporting that residual alterations remain following recovery.

Keywords: Low back injury; Neuromuscular patterns; Motion characteristics; Pain behaviors

Introduction

Recurrent injuries contribute disproportionately to high costs associated with low back pain (LBP) (Wasiak et al., 2006). Published guidelines indicate that most LBP will resolve itself (Chou et al., 2007) but while 90% of low back injuries are reported to resolve themselves with respect to pain, 62% report pain after 1 year making a strong argument that low back injury (LBI) should not be left to resolve itself (Hestbaek et al., 2003). Few objective physiological or functional measurements are used to assess recovery with self-reported measures of pain and function along with functional assessments most frequently used to assess recovery and the likelihood of returning to work (Reme et al., 2009; Du Bois et al., 2009).

Theoretical (Panjabi, 2006) and modeling work (Cholewicki and McGill, 1996) has linked trunk neuromuscular alterations to mechanical spinal instability and LBI. Increased agonist and antagonist co-activation has been associated with active stiffness enhancing spinal stability (Kavcic et al., 2004, Granata and Orishimo, 2001 and Cholewicki and McGill, 1996), with an altered response from even one muscle site influencing stability (Kavcic et al., 2004). Altered surface electromyographic (EMG) recordings have been reported for individuals with LBP (van Dieen et al., 2003, Silfies et al., 2005 and Hubley-Kozey and Vezina, 2002). A meta-analysis in 2005 concluded that EMG techniques have the potential to serve as a marker for LBP (Geisser et al., 2005), but most studies have only examined chronic LBP i.e. those with pain that has lasted for more than 3 or 6 months. Complexities of chronic LBP with respect to pain-related psychological factors (Sullivan et al., 2006) as well as central nervous system changes (Arendt-Nielsen et al., 1994 and Nie et al., 2005) limit the ability to extrapolate findings to those who experience episodic LBP i.e. those with more than 1 month of pain free time between episodes (Stanton et al., 2009). Altered neuromuscular responses have been reported with experimental pain models in healthy asymptomatic individuals using biochemical agonists (Hodges et al., 2003) or inducing discomfort by prolonged activities (Gregory and Callaghan, 2008). Collectively these studies provide evidence that neuromuscular responses are altered in chronic LBP and with induced acute pain, but minimal work has been done on whether alterations are apparent after remission of symptoms in episodic cases.

MacDonald et al. examined participants with multiple recurrent LBP episodes during a period in which participants had no pain demonstrating altered onset times and amplitudes in lumbar

multifidus during rapid arm movement (MacDonald et al., 2009) and trunk loading tasks compared to asymptomatic controls. The authors suggested that these altered multifidus patterns may leave the spine vulnerable to repeat episodes. These findings provide evi dence of residual dysfunction. However, experimental and biome chanical modeling evidence suggests that all trunk muscles are important for spinal function and stability (Cholewicki and VanV liet, 2002; Kavcic et al., 2004). Furthermore, different segments within a muscle can respond differently to external perturbations (Butler et al., 2009a,b; Jonsson, 1973; Mirka et al., 1997; Vink et al., 1988) supporting the need to examine the interactions among a comprehensive set of trunk muscle sites. Given the high incidence of low back re injury, whether these residual alterations to trunk muscle activation patterns remain during recovery could help to explain low back re injury. As return to work and clinical decisions are made within the sub acute (typically within 4 12 weeks after the event) phase of a LBI, determining whether al tered patterns are present in that phase and whether they exist in a comprehensive set of trunk muscles would further our under standing of LBI recovery and perhaps provide evidence for clinical management decisions.

Our previous work reported on a comprehensive set of abdom inal and back extensor muscle sites, illustrated distinctive activa tion amplitude patterns in response to a highly controlled task in a healthy population (Butler et al., 2009a). The present study sought to determine if neuromuscular amplitude patterns were different in a group within the sub acute phase of a LBI/pain epi sode that were deemed recovered compared to controls. Multivar iate techniques tested the hypothesis that differences exist in the characteristics of the neuromuscular strategies between the two groups despite similar task performance.

2. Methods

2.1. Sample

This prospective comparative study included 54 asymptomatic (57% women) and 33 LBI (60% women) subjects between the ages of 20 55 years. Controls were recruited from local advertisements and had no history of LBI or pain during the past year or a LBP epi sode that resulted in missing time from work or for which they sought medical attention. LBI participants were recruited from physiotherapy clinics and advertisements. They reported an epi sode of "pain between the lower ribs and gluteal folds" (Spitzer et al., 1987) that was associated with a mechanical event based on history but that did not have a specific cause such as fracture or other disease processes (Waddell et al., 1992). LBI participants were tested within 12 weeks post injury (sub acute phase). They were deemed recovered based on self reported remission of symp toms and resumption of normal activities or were within one week of returning to these activities. Inclusion/exclusion were deter mined through a health screening questionnaire and standard physiotherapy assessment. Participants signed an informed con sent approved by the Health Sciences Research Ethics Board, Dal housie University.

Mass, height, waist girth and maximum reach were measured (Butler et al., 2009a). Age, sex and physical activity levels were re corded. An abdominal test graded as normal, 60 or 80 percent of normal function was used (Kendall and McCreary, 1983). As part of the physiotherapy assessment posture (including scoliosis and kyphosis), neurological testing including reflexes (patellar and Achilles tendon, hamstrings), myotomes and dermatomes along with clinical instability tests (Mens et al., 2001; Albert et al., 2000; Kasai et al., 2006; Vleeming et al., 2002; Ostgaard et al., 1994; Hicks et al., 2005; Hicks et al., 2003) were conducted on LBI group (Table 1). Pain intensity before and after testing was as sessed using a visual analog scale (VAS; 0 mm = no pain and 100 mm = extreme pain) (Wewers and Lowe, 1990). Roland Morris Questionnaire (Roland and Morris, 1983) was used to assess low back related disability and the Pain Catastrophizing Scale to assess catastrophic thinking related to pain (Sullivan et al., 1995). Finally, pain behaviors were identified from videotaped recordings of facial expressions and body behaviors during experimental tasks (Sullivan et al., 2006).

2.2. Sensor Placement

Surface electrodes (Meditrace, pre gelled, Ag/agCl 0.79 cm^2, 30 mm inter electrode distance) were placed in a bipolar configu ration over 12 bilateral trunk muscle sites (right (R) and left (L) sides of the body) based on standard placements as illustrated in Fig. 1 and adjusted based on individual anthropometrics (Butler et al., 2009a). Abdominal sites included: lower (LRA midpoint be tween the pubis symphysis and umbilicus) and upper rectus abdo minis (URA midpoint between the umbilicus and the sternum); anterior (EO1 over the eighth rib), lateral (EO2 approximately 15 cm lateral to the umbilicus at a 45° angle) and posterior fibers (EO3 halfway between the iliac crest and lower portion of the rib cage) of external oblique and internal oblique (IO centered in the triangle formed by the inguinal ligament, lateral border of rectus sheath and the line between the two anterior superior iliac spines). Six bilateral back extensor sites included: lumbar erector spinae at L1 and L3 at 3 and 6 cm from the midline to represent the longiss imus and iliocostalis muscle sites, respectively (L13, L16, L33, L36); quadratus lumborum at L4 at approximately 8 cm from the mid line (L48); and multifidus at L5 at 1 2 cm from the midline (L52). Submaximal validation exercises were performed.

Angular motion of the trunk and pelvis was monitored using a Flock of Birds™ (FOB) motion system (Ascension Technology Inc., Burlington, Vermont) throughout the task. Two electromagnetic sensors were placed over the spinous process at 7th thoracic verte brae (trunk) and over the left iliac crest (pelvis).

2.3. Experimental trials

Subjects stood at a table (adjusted to standing elbow height) and performed three trials of lifting and replacing a 2.9 kg load using both hands in two reach conditions (Fig. 2) while minimizing trunk and pelvis motion (Butler et al., 2009a). Subjects were re quired to move the load 4 5 cm off the table in a controlled man ner and lower within a standardized 3 s count. An event marker identified lift, transition and lowering phases. Only the lift phase was examined given similar patterns were found for the two other phases (Butler et al., 2009a). If trunk or pelvis motion was visible during the trial or upon review if the any of the three angular dis placement traces exceeded 3°, the trial was repeated.

Table 1
Clinical instability tests.

Aberrant movement during lumbar spine flexion/ extension	Hicks et al. (2005)
Passive straight leg raise range of motion	Hicks et al. (2005)
Posterior-to-anterior mobility testing	Hicks et al. (2005)
Prone instability test	Hicks et al. (2005)
Posterior pelvic pain provocation test	Ostgaard et al. (1994)
Provocation of long dorsal sacroiliac ligament	Vleeming et al. (2002)
Active straight leg raise test	Mens et al. (2001)
Provocation of pubic symphysis with palpation	Albert et al. (2000)
Modified trendelenburg test	Albert et al. (2000)
Prone leg extension	Kasai et al. (2006)

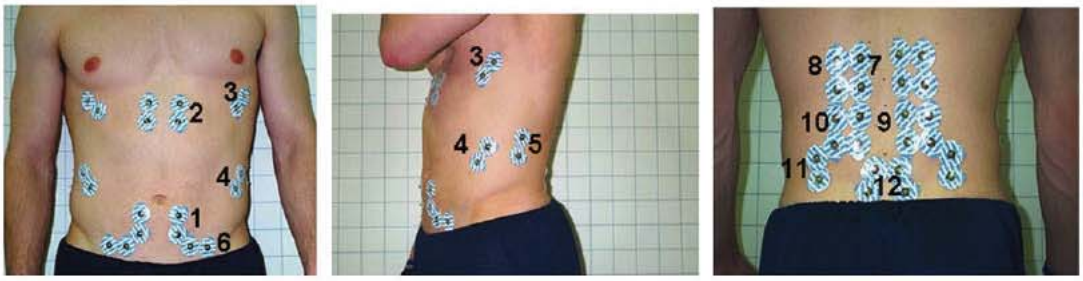

Fig. 1. Muscles sites on right and left sides of the body. 1 = LRA; 2 = URA; 3 = EO1; 4 = EO2; 5 = EO3; 6 = IO; 7 = L13; 8 = L16; 9 = L33; 10 = L36; 11 = L48; 12 = L52.

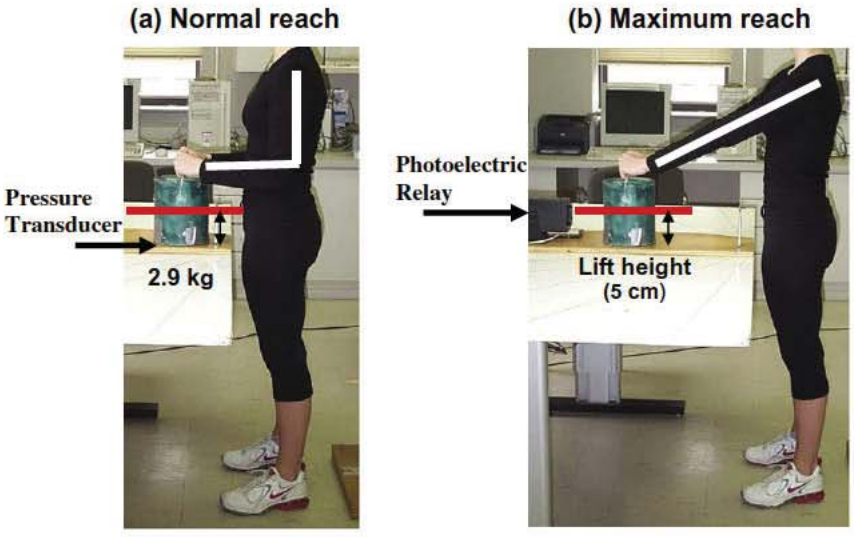

Fig. 2. Experimental set-up and subject posture (a) normal and (b) maximum reach.

2.4. Electromyographic (EMG) normalization

Post experimental trials, eight different maximum voluntary isometric contraction (MVIC) exercises were performed (Butler et al., 2009a). These included a supine sit up; sitting axial rotation to the right to left; side lying lateral flexion to the right and left with contralateral hip hike; prone back extension and back exten sion coupled with axial rotation to the right and to the left. During each exercise standardized verbal encouragement and feedback was provided to ensure maximum effort and correct performance. To avoid fatigue subjects were given at least a 2 min rest period between trials.

2.5. Data acquisition and processing

The event marker system synchronized the EMG and FOB data. Raw EMG signals were pre amplified (500×) and further amplified using three AMT 8 EMG systems (bandpass 10 1000 Hz, CMRR = 115db, input impedance 10 GΩ, Bortec Inc., Calgary, Alber ta). EMG and event signals were sampled at 1000 Hz using a 16 bit analog to digital converter (National Instruments, CA 1000) using LABVIEW™ and stored on a personal computer. FOB signals were sampled at 50 Hz using a 12 bit analog to digital converter (Na tional Instruments, CA 1000) using LABVIEW™.

Raw EMG signals were filtered using a recursive 5th order But terworth 30 Hz high pass filter to remove the ECG artifact (Butler et al., 2009c) and then an inverse FFT filter was applied to remove low level noise from the FOB system. Root mean square (RMS) amplitude was calculated for the lift phase. Maximum RMS amplitude from the normalization exercises was used to normalize the lift phase RMS amplitude to a percentage of MVIC (%MVIC) (Vezina and Hubley Kozey, 2000). Three trials were averaged for each subject and condition.

For the FOB data processing, three dimensional angular data was filtered at 1 Hz with a recursive 2nd order Butterworth filter and maximal angular displacement was calculated for yaw, pitch and roll of the trunk and pelvis for the lift phase. The measured angular data were obtained with respect to a global reference but correspond to lateral bend (yaw), flexion extension (pitch), axial rotation (roll) in an anatomical reference system consistent with previous methods (Silfies et al., 2009).

2.6. EMG data analyses

The analytical process using pattern recognition algorithms is outlined in Fig. 3 with more detailed descriptions found elsewhere (Butler et al., 2009a). For the present study the data matrix ($X_{[n,p]}$) consists of amplitude patterns where $n = 174$ [87 subjects × 2 reaches] and $p = 24$ muscle sites (Fig. 3a). Briefly, an eigenvector decomposition was applied to the cross product matrix of the data matrix $X_{[n,p]}$. (Fig. 3b). Principal patterns (PPi) were extracted rep resenting the different features from the amplitude pattern (Fig. 3c). These features reduce the data and are mathematically derived directly from the measured amplitude patterns capturing common patterns observed in the data matrix that allow for both the overall amplitude and for relative changes in amplitudes among the 24 muscle sites to be captured. Finally, a weighting coefficient (PP_i *score)* was calculated for each feature (PP) that

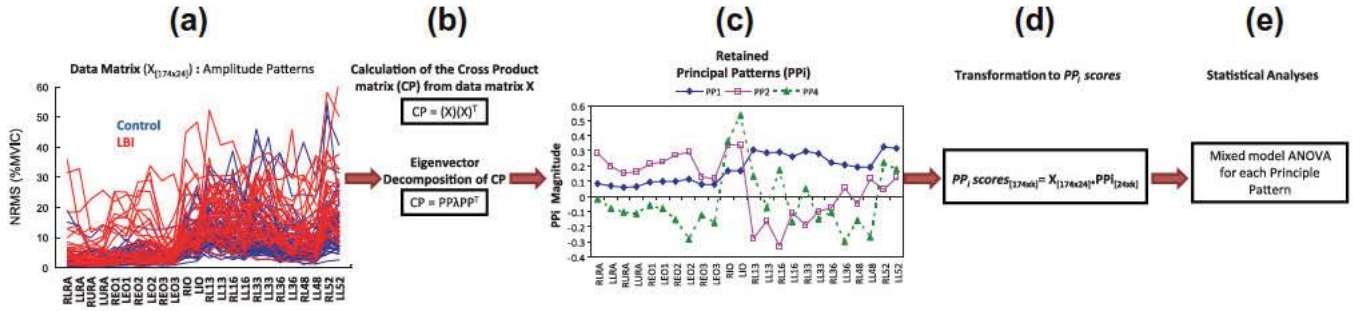

Fig. 3. (a) The amplitude patterns for control (blue) and LBI (red) subjects make up the data matrix (X). (b) The Cross Product matrix is then calculated from the data matrix (X) and subsequently undergoes eigenvector decomposition. (c) The resulting transform matrix consisted of orthogonal principal patterns (PPi) that captured the features from the measured amplitude patterns. (d) Only those principal patterns (k) that explained 95% of the total variation in the data were retained for interpretation. These k patterns transformed the measured amplitude patterns to a reduced number of uncorrelated variables (PP_i *scores*), which were used in statistical analyses (e).

depicts how much that feature contributes to the measured pat tern (Fig. 3d). These *scores* subsequently were used for statistical analyses (Butler et al., 2009a) (Fig. 3e).

Patterns capturing up to 95% of the variance were retained for further analysis (Fig. 3C). Hence PCA reduced the data with the fea tures derived from the data itself. Reconstructions using k principal patterns were performed (mean *score* for each group and reach condition) and reconstructed amplitude patterns in %MVIC were graphically compared to measured amplitude patterns to illustrate how well the salient features were captured (Hubley Kozey and Smits, 1998). The mean pattern from a subsample of 4 5 measured patterns that correspond to both high and low *scores* were dis played to aid in the interpretation of the activation amplitude fea ture captured for each PP. This was a two step process. The subsample amplitude patterns was first selected based on similar PP1 mean *scores* since different *scores* can notably influence its magnitude making interpretation of the feature more difficult. Sec ond, high or low *scores* were identified for a given PPi that also had *scores* close to zero for the remaining PPs. To highlight the PP2 fea ture, for example, the amplitude patterns that corresponded to high or low PP2 *scores* that had a mean PP1 *score* and a zero value for PP3 and PP4 *scores* would be included in the subsample (Butler et al., 2009a).

2.7. Statistical analysis

For each analysis of variance (ANOVA) model, assumptions of normality and homogeneity of variance were examined. Mixed model ANOVAs tested for group and condition differences in PP scores ($\alpha = 0.05$). All statistics were calculated using Minitab™ (Minitab Inc., State College, PA. Version 14). When applicable, post hoc analyses were performed using Bonferroni corrections.

3. Results

Descriptive statistics are in Table 2. The LBI group was older with greater BMI and waist circumference measures ($p < 0.05$) than controls. Abdominal function grades were similar between groups, with control group reporting on average one more bout of aerobic activity and one less abdominal training session per week than LBI group (Table 2). LBI subjects were tested 6.5 (±3.0) weeks post pain episode. They reported minimal pain before and after the test session, low catastrophizing and disability scores (Table 3) with no pain behaviors displayed. Information on previous LBP episodes are in Table 3 with time from previous episode to current injury greater than 3 months for the majority supporting the episodic nature of the injury. The physiotherapy assessment showed that 94% of LBI subjects had no sensory deficit with 2 having one minor

Table 2
Mean (standard deviation) and statistical results for subject demographics and number (percent) of individuals for each abdominal function level.

Variable	Asymptomatic	LBI	p-Value
Age (years)	31.9(8.7)	40.2 (12.2)	0.001*
Mass (kg)	71.6(15.1)	79.1 (21.9)	0.092
Height (cm)	171.0(8.9)	169.7(9.2)	0.517
BMI (kg/m^2)	24.4 (4.0)	27.3 (6.4)	0.025*
Waist girth (cm)	80.2(11.2)	87.7(17.4)	0.037*
Aerobic activity (times/week)	3.5 (3.0)	2.3(2.1)	
Abdominal training (times/week)	1.6(2.4)	2.5 (2.7)	
Abdominal function test			
Normal	44 (83%)	23 (70%)	
80%	3 (6%)	7 (21%)	
60%	6(11%)	3 (9%)	

* $p < 0.05$.

Table 3
Mean (standard deviation) for pain related variables and percentage of subjects for injury history variables.

Variable	LBI
VAS (before)	17.1 (19.1)
VAS (after)	18.3 (20.9)
Roland Morris disability scale	4.2 (4.3)
Pain catastrophizing scale	11.6 (9.3)
Years from first LBI[a]	
<1 years	26%
1–4 years	30%
>10 years	44%
Number of previous injuries	
0 – First time	15%
1–4 Injuries	52%
5–10 Injuries	21%
>10 Injuries	12%
Time from previous episode	
0 – First time	15%
1–3 months	18%
3–11 months	21%
1 year or more	46%

[a] Six subjects could not recall date of first injury.

deficit and 9 classified as having segmental instability (Stuge et al., 2004).

3.1. Motion

Maximum range trunk and pelvis motion in any one direction was less than 1° for both groups and in both reaches (Table 4). Thus the major external moment acting on the spine was due to the external load. Given the condition restraints and that the reach val

Table 4
Maximum rotational motion for the two sensors in degrees with mean (standard deviation).

Condition	Group	Trunk			Pelvis		
		Lateral bend	Flexion–extension	Axial rotation	Lateral bend	Flexion–extension	Axial rotation
Normal	Asymptomatic	0.23 (±0.17)	0.50 (±0.44)	0.56 (±0.50)	0.19 (±0.12)	0.37 (±0.30)	0.31 (±0.24)
	LBI	0.19 (±0.11)	0.35 (±0.23)	0.39 (±0.33)	0.20 (±0.17)	0.14 (±0.11)	0.29 (±0.19)
Maximum	Asymptomatic	0.36 (±0.18)	0.68 (±0.43)	0.65 (±0.45)	0.26 (±0.21)	0.47 (±0.39)	0.36 (±0.21)
	LBI	0.26 (±0.18)	0.81 (±0.78)	0.61 (±0.44)	0.33 (±0.31)	0.32 (±0.21)	0.42 (±0.32)

Note 15% of subjects were missing all of the motion due to technical issues following testing.

ues were similar between groups; we assumed similar moments of force in the sagittal plane during normal and maximum reach con ditions between the groups.

3.2. EMG amplitude pattern analysis

Measured amplitude patterns for normal and maximum reach are in Fig. 4. Patterns showed low abdominal and higher back extensor activation with higher overall amplitudes in maximum reach (4b). While amplitude patterns were similar, subtle differ ences between reaches and groups were observed. Most notably LBI subjects showed higher overall amplitudes compared to con trols with a distinctive drop in bilateral EO3 activity. Overall LBI group variability was higher than controls.

Four principal patterns explained 95% of the total variation in measured data (Fig. 5). Mean measured amplitude patterns and reconstructed patterns using four principal patterns for both groups and reach conditions are in Fig. 4. Reconstructed patterns confirm that four patterns captured the salient features observed in the measured data.

PP1 captured the dominant shape and magnitude differences among abdominal and back extensor muscle sites (Fig. 5a shape/ magnitude feature); low abdominals and higher back extensor amplitudes with lower activity at EO3 site and higher IO amplitudes than the other abdominals. Magnitude differences are illustrated by high and low PP_1 *scores* (Fig. 5b). There was a significant group by condition ($p < 0.05$) interaction with post hoc results indicated in Table 5. Compared to controls, LBI subjects had significantly higher PP_1 *scores* for both reaches ($p < 0.01$). For both groups, PP_1 *scores* were significantly higher in maximum reach than normal reach ($p < 0.01$) with a greater increase in magnitude for LBI sub jects in maximum reach (see Fig. 4b). Trunk muscle sites on average were 4.7% and 5.6% MVIC higher for LBI individuals in normal and maximum reach, respectively.

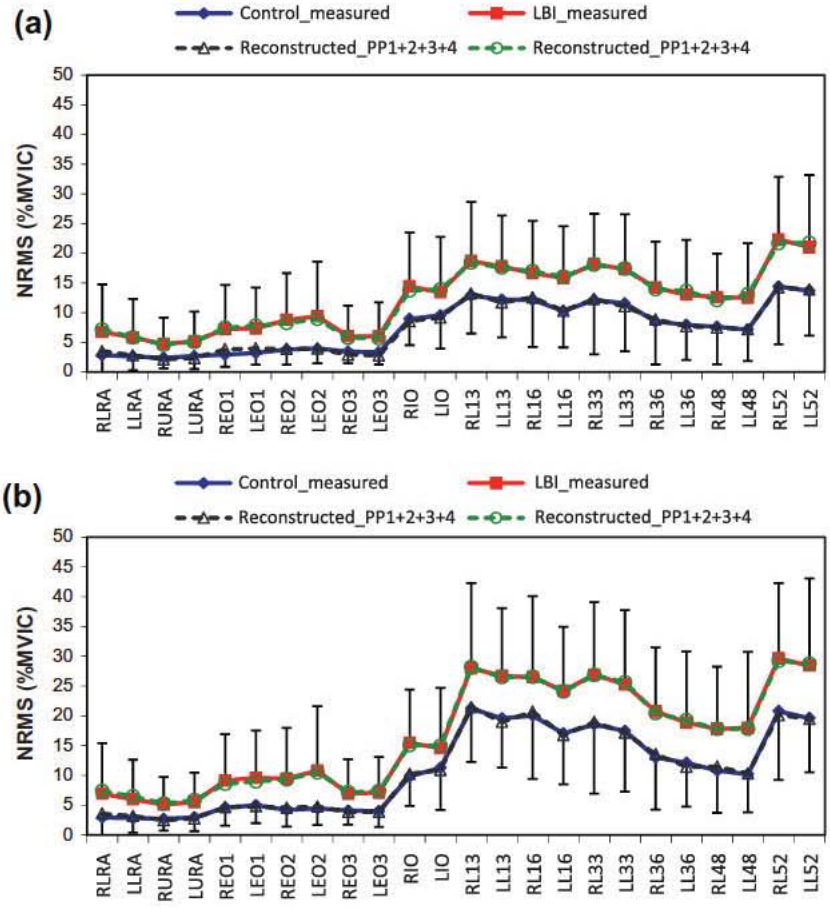

Fig. 4. Mean (standard deviation) measured and reconstructed activation amplitude patterns comparing control and LBP groups during the lift phase for (a) *normal* and, (b) *maximum* reach. The *y*-axis is %MVIC and the *x*-axis is muscle sites. Each panel includes asymptomatic reconstructed (△), LBP reconstructed (○), asymptomatic measured (◇) and LBP measured (□) amplitude patterns. Adjoining lines are for illustrative purposes only.

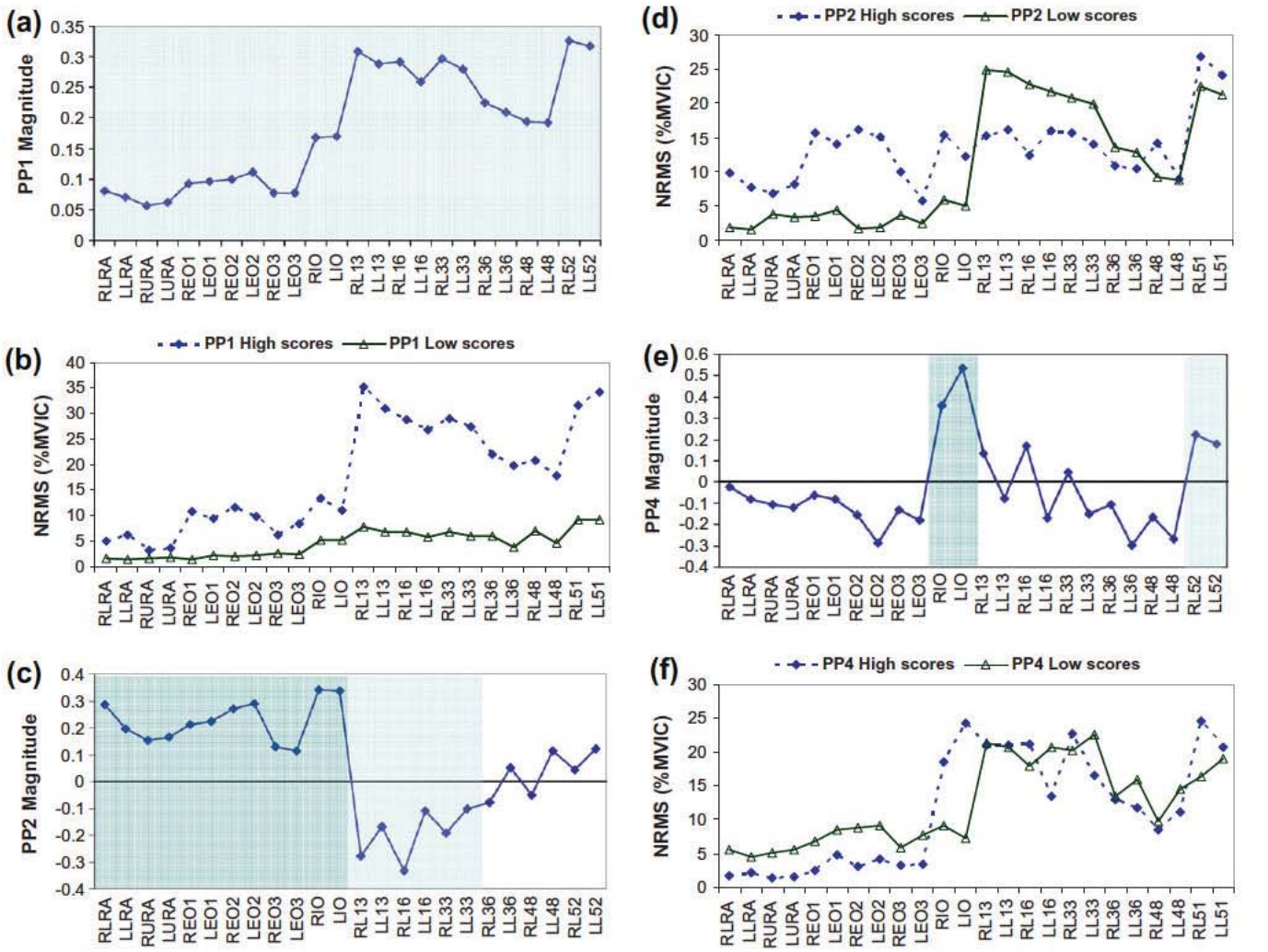

Fig. 5. PP1 accounted for 88.2% of the variability whereas PP2, PP3 and PP4, captured 3.1%, 2.1% and 1.7%, respectively of the total variance reflecting different amplitude recruitment strategies during the lift/replace task. Shaded areas illustrate the portion of the pattern where the greatest variance was explained by associated PPs. PP1 (a), PP2 (c) and PP4 (e) had significant group effects. Below each pattern b, d and f depict high and low *scores* for each pattern. (b) high PP_1 *scores* depicts the overall higher magnitude in both abdominals and back extensors, (d) high PP_2 *scores* depict the higher abdominal compared to back extensor and (f) high PP_4 *scores* illustrate an increase in IO activity compared to the other abdominals and to a lesser extent an increase in MT.

Table 5
Statistical post hoc results and mean and standard deviation *for PPj scores.*

Reach	Group	*PPi scores*	*PP2 scores*	*PP3 scores*	*PP4 scores*
Normal	Asymptomatic	41.7 (20.7)*	3.1 (11.8)*	−0.26 (9.4)	3.4 (5.3)
	LBI	65.2(29.3)*,**			1.0 (12.3)*,**
Maximum	Asymptomatic	62.1 (25.4)	−2.9 (13.5)	−1.2 (11.6)	3.4 (7.0)
	LBI	91.2 (37.4)**			−1.0 (13.9)**
	Asymptomatic LBI		−2.0 (8.4)		
			3.4 (17.8)**		

Interaction effect for PP1 and PP4 ($p < 0.05$).
* Significantly different from maximum reach ($p < 0.0125$).
** Significantly different from asymptomatic controls ($p < 0.0125$).

Given that the patterns are linear combinations, opposite polarity between abdominal and the majority of the back extensor sites in PP2 depicts a relationship where agonist muscles have a decrease in amplitude with respect to PP1 and the antagonist muscle groups have an increase in amplitude (co activation feature) (Fig. 5c). High positive PP_2 *scores* capture increased abdominals with the lowest response for EO3 sites while the majority of the back extensor sites had a reduction in the amplitude relative to their PP1 value (i.e. PP1 times PP_1 *score*). Significant main effects were found for group ($p < 0.05$) and condition ($p < 0.05$). LBI subjects had significantly higher PP_2 *scores* compared to controls (Table 5) indicating that the LBI group had greater relative increases in abdominal site activation in general (EO3 had a smaller relative increase compared to the other abdominal sites) resulting in a smaller relative difference in activation amplitudes between abdominals and back extensor sites (more co activity see

Fig. 5d). Significantly higher PP_2 *scores* were found in normal compared to maximum reach ($p < 0.02$) indicating that during maximum reach in both groups back extensors had a higher relative increase in amplitudes compared to the increase in abdominal sites.

PP3 had a significant condition main effect only ($p < 0.05$). PP_3 *scores* were significantly higher in normal reach compared to maximum reach. Given that condition did not interact with group and that interpretation of this feature has been presented elsewhere (Butler et al., 2009a), it was not further described in this paper.

PP4 captured a selective increase in amplitude for internal oblique and to a lesser extent multifidus muscle sites (local muscle synergism feature, Fig. 5e). There was a significant group by condition interaction ($p < 0.05$). All pairwise comparisons were significant ($p < 0.01$) except between reaches in controls ($p > 0.01$) (Table 5). For LBI subjects, PP_4 *scores* were lower ($p < 0.01$) and close to zero compared to controls indicative that this pattern contributed minimally to the LBI group. The effect of this feature is illustrated by high and low *scores* (Fig. 5f) and in part by the relative amplitude of internal oblique to all other abdominal muscle sites and the multifidus to all other back extensor muscle sites. For example in the controls the ratios of IO to all abdominals and MT to all back extensors was relatively consistent between reaches for the controls (IO 3.0, MT 2.1 for normal and IO 2.8, MT 2.0 for maximum) but drops for the LBI group (IO 2.8, MT 2.0 for normal and IO 2.0,MT 1.8 for maximum). This selective recruitment was less pronounced for back extensor sites and multifidus.

4. Discussion

Trunk muscle activation amplitude patterns differed between controls and individuals who self reported recovery following a LBI/pain episode. Low pain levels, minimal functional limitations and no pain behaviors support their perception of recovery. The task was highly controlled so differences could not be explained by timing, motion and task demand differences between groups. Whether individuals had not recovered, whether adaptive response to the residual effects of the injury still existed or whether these patterns were pre existing cannot be established for certain, however, specific patterns are different between the controls and LBI group.

Higher percentage of maximal activation of trunk muscles in LBI subjects (PP1) could be explained by muscle strength deficits consistent with previous findings of lower strength values for chronic LBP participants (Cassisi et al., 1993). A lower percentage of LBI subjects had normal abdominal function grades compared to controls (Table 2), but we did not record muscle strength which is a limitation. If the same level of active stiffness was needed by both groups to maintain stability and perform this task, then the LBI group needed more activity to produce the required force. However if passive stiffness was decreased consistent with alteration in passive structures as per the three model subsystem (Panjabi, 2006), then the increase activity could be responding to this need for more active stiffness. While the ability to produce MVIC amplitudes during normalization exercises has been questioned for individuals who are experiencing pain (Marras and Davis, 2001) the low VAS pain scores, minimal VAS pre and post test change, low pain catastrophising scores, and no pain behaviors in the LBI group do not support this explanation for higher amplitudes. Normalization procedures are required with those based on MVIC considered a reproducible standard (Burden, 2010) for asymptomatic subjects and while there are no studies on the trunk musculature, evidence from the knee arthritis literature illustrates that both those with knee arthritis pain and healthy voluntarily activate their quadriceps muscles to similar percentages of their stimulated maximum (Lewek et al., 2004). Furthermore decreased MVIC capabilities would not explain lower activation for posterior fibers of the external oblique with respect to the anterior and lateral fibers in the LBI group whereas in the control group all external oblique fibers were at similar activation levels. Thus working at a higher percentage of their maximum for the LBI group to perform a similar task increases the potential for muscle fatigue and over time increase the risk for a spinal instability related injury. The differences between groups within a muscle or between conditions as well as the relative differences among muscle sites were not uniform. The PCA results allowed for comparing these relative differences by picking out key features from the amplitude pattern data that reduced the large set of data into 3 additional patterns (PP2 PP4) that we would not have established apriori.

Higher relative abdominal and back extensor activation was found (PP2) in the LBI group whereas the controls had higher back extensor amplitudes relative to abdominal muscles. The difference was greater during maximum lift illustrating increased agonist activity in response to the higher external demand whereas the antagonist abdominals remained under 5% MVIC for controls (except IO sites), a level deemed appropriate for maintaining joint stability (Cholewicki et al., 1997). Higher agonist antagonist co activation is a strategy shown to increase active stiffness of the spine (Tucker and Hodges, 2009), to enhance spinal stability in response to fatigue (Granata et al., 2004) or to reduced stability conditions (Granata and Orishimo, 2001). Increased co activation in the LBI group could also reflect a guarding response (van der Hulst et al., 2010) associated with pain. Our results do not support one explanation over the other for increased co activation as pain scores were low and only one third of LBI participants had clinical instability (Stuge et al., 2004). While this co activation adaption may be a short term solution to prevent aggravation of tissues, pain or movement related injury, there may be long term consequences from increased loading and reduced motion. Furthermore, recent evidence supports that trunk flexor extensor co activation is a precursor to developing LBP in asymptomatic individuals (Nelson Wong and Callaghan, 2010).

A novel finding was lower relative activation of posterior fibers of external oblique in the LBI group as illustrated in Figs. 4 and 5 (PP1 and PP2) compared to other external oblique fibers. This finding cannot be explained by decreased strength or inhibition during MVIC testing, but lower activation supports a reduced neural drive to the posterior more vertically oriented external oblique fibers in the LBI group. Altered posterior external oblique temporal activation patterns were found in asymptomatic individuals who were unable to stabilize their pelvis during a leg loading task compared to those who could (Hubley Kozey et al., 2010). Furthermore the group that could stabilize their pelvis recruited all of their abdominals to similar amplitudes for the most demanding task (Davidson and Hubley Kozey, 2005), a strategy referred to as abdominal bracing. Abdominal bracing has been suggested as a strategy to increase spinal stability (Brown et al., 2006) with the theory that even one inappropriate muscle response can disrupt stability (McGill, 2002). Thus reduced neural drive to the posterior external oblique fibers may contribute to an unstable environment of a spinal unit.

While the importance of local versus global muscles in spinal stability and LBI management has been debated, the consensus is that all trunk muscles are important (Kavcic et al., 2004). Delayed timing in deep local muscles; multifidus and transverse abdominis/internal oblique with chronic LBP (Hodges and Richardson, 1996) and in short fibers of the multifidus in those in remission from an episode of LBP (MacDonald et al., 2009) have been reported. Local abdominal (IO) muscle sites showed greater relative increase compared to all other abdominal sites in response to higher physical demands for the controls but not for the LBI group per

haps indicative of impairment in local muscle responsiveness in the LBI subjects. These findings are consistent with the local mus cle alterations previously reported in chronic LBP and acute pain models including inhibition, unloading/substitution/decreased sensory function (Hides et al., 1996; Claeys et al., 2011). However, large variation in PP4 scores indicates that the LBI group included individuals with inhibited as well as enhanced activation in local muscles, suggesting that there are potential subgroups. This may have implications for therapeutic interventions in that those with enhanced local activity may not benefit from therapies that focus on selectively activating deep muscles. Thus our results provide evidence of local muscle alterations although it is the first time that these impairments are reported during a functional but highly controlled task in those recovered from an episode of LBI.

While our LBI sample was older and had greater waist circum ference and BMI measures than controls, the age difference was less than a decade and differences in muscle activation patterns would not be expected. Increased mass and adipose tissue would decrease raw amplitudes but normalization procedures address this issue. The highly controlled task examined reduces the general applicability of the findings but was necessary to eliminate the ef fects that alterations in motion and timing found in those experi encing LBP (Silfies et al., 2009) could have on neuromuscular responses. Finally, the LBI group was homogeneous with respect to inclusion criteria, but variability in measured amplitudes in % MVIC and principal patterns *scores* were higher in the LBI group. Higher variability reflects heterogeneity which could relate to loca tion of injury/pain and clinical assessment. Perhaps examining the relationship between pain locations and specific alterations in neu romuscular responses could provide additional objective evidence to support classification methods presently being developed (Fritz et al., 2007). The results also support that LBI management ap proaches should focus on the trunk musculature unit and interac tions among these muscles. Targeting specific muscles for selective recruitment training should be based on objective outcomes that illustrate an inhibition exists in the target muscles as our results show that the abdominal and back extensors work as a unit with distinctive pattern alterations in muscles other than just deep trunk muscles. Finally further study could determine whether these altered neuromuscular responses can predict reoccurrence by increasing our understanding of recovery.

In conclusion, differences exist in activation *amplitude patterns* between controls and those within the sub acute phase of a LBI who were deemed recovered. PCA identified specific features from the amplitude data that can now be used to develop standardized measures. Specifically, an overall increase in activity of abdominals and back extensors, increased agonist antagonist co activation strategy, reduced posterior oblique fiber activation and impaired local muscle responses to increased demand was found in the LBI group. These findings support that alterations exist despite no pain or functional limitations and hence have the potential to provide an objective physiological assessment of recovery.

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References

Albert H, Godskesen M, Westergaard J. Evaluation of clinical tests used in classification procedures in pregnancy-related pelvic joint pain. Eur Spine J 2000;9(2):161–6.

Arendt-Nielsen L, Brennum J, Sindrup S, Bak P. Electrophysiological and psychophysical quantification of temporal summation in the human nociceptive system. Eur J Appl Physiol 1994;68(3):266–73.

Brown SH, Vera-Garcia FJ, McGill SM. Effects of abdominal muscle coactivation on the externally preloaded trunk: variations in motor control and its effect on spine stability. Spine 2006;31(13):E387–93.

Burden A. How should we normalize electromyograms obtained from healthy participants? What we have learned from over 25 years of research. J Electromyogr Kinesiol 2010;20(6):1023–35.

Butler HL, Hubley-Kozey CL, Kozey JW. Electromyographic assessment of trunk muscle activation amplitudes during a simulated lifting task using pattern recognition techniques. J Elctromyogr Kinesiol 2009a;19(6):e505–512.

Butler HL, Hubley-Kozey CL, Kozey JW. Activation amplitude patterns do not change for back muscles but are altered for abdominal muscles between dominant and non-dominant hands during one-handed lifts. Eur J Appl Physiol 2009b;106(1): 95–104.

Butler HL, Newell R, Hubley-Kozey CL, Kozey JW. The interpretation of abdominal wall muscle recruitment strategies change when the electrocardiogram (ECG) is removed from the electromyogram (EMG). J Electromyogr Kinesiol 2009c;19(2):e102–113.

Cassisi JE, Robinson ME, O'Conner P, MacMillan M. Trunk strength and lumbar paraspinal muscle activity during isometric exercise in chronic low-back pain patients and controls. Spine 1993;18(2):245–51.

Cholewicki J, McGill SM. Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. Clin Biomech 1996;11(1): 1–15.

Cholewicki J, VanVliet JJ. 4th Relative contribution of trunk muscles to the stability of the lumbar spine during isometric exertions. Clin Biomech 2002;17(2): 99–105.

Cholewicki J, Panjabi MM, Khachatryan A. Stabilizing function of trunk flexor–extensor muscles around a neutral spine posture. Spine 1997;22(19):2207–12.

Chou R, Qaseem A, Snow V, Casey D, Cross Jr JT, Shekelle P, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med 2007;147(7):478–91.

Claeys K, Brumagne S, Dankaerts W, Kiers H, Janssens L. Decreased variability in postural control strategies in young people with non-specific low back pain is associated with altered proprioceptive reweighting. Eur J Appl Physiol 2011; 111(1):115–23.

Davidson KL, Hubley-Kozey CL. Trunk muscle responses to demands of an exercise progression to improve dynamic spinal stability. Arch Phys Med Rehabil 2005;86(2):216–23.

Du Bois M, Szpalski M, Donceel P. Patients at risk for long-term sick leave because of low back pain. Spine J: Off J North Am Spine Soc 2009;9(5):350–9.

Fritz JM, Cleland JA, Childs JD. Subgrouping patients with low back pain: evolution of a classification approach to physical therapy. J Orthopaed Sports Phys Ther 2007;37(6):290–302.

Geisser ME, Ranavaya M, Haig AJ, Roth RS, Zucker R, Ambroz C, et al. A meta-analytic review of surface electromyography among persons with low back pain and normal, healthy controls. J Pain 2005;6(11):711–26.

Granata KP, Orishimo KF. Response of trunk muscle coactivation to changes in spinal stability. J Biomech 2001;34(9):1117–23.

Granata KP, Slota GP, Wilson SE. Influence of fatigue in neuromuscular control of spinal stability. Hum Factors 2004;46(1):81–91.

Gregory DE, Callaghan JP. Prolonged standing as a precursor for the development of low back discomfort: an investigation of possible mechanisms. Gait Posture 2008;28(1):86–92.

Hestbaek L, Leboeuf-Yde C, Manniche C. Low back pain: what is the long-term course? A review of studies of general patient populations. Eur Spine J 2003;12(2):149–65.

Hicks GE, Fritz JM, Delitto A, Mishock J. Interrater reliability of clinical examination measures for identification of lumbar segmental instability. Arch Phys Med Rehabil 2003;84(12):1858–64.

Hicks GE, Fritz JM, Delitto A, McGill SM. Preliminary development of a clinical prediction rule for determining which patients with low back pain will respond to a stabilization exercise program. Arch Phys Med Rehabil 2005;86(9): 1753–62.

Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine 1996;21(23):2763–9.

Hodges PW, Richardson CA. Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine 1996;21(22):2640–50.

Hodges PW, Moseley GL, Gabrielsson A, Gandevia SC. Experimental muscle pain changes feedforward postural responses of the trunk muscles. Exp Brain Res 2003;151(2):262–71.

Hubley-Kozey CL, Smits E. Quantifying synergist activation patterns during maximal plantarflexion using an orthogonal expansion approach. Hum Mov Sci 1998:17347–65.

Hubley-Kozey CL, Vezina MJ. Differentiating temporal electromyographic waveforms between those with chronic low back pain and healthy controls. Clin Biomech 2002;17(9–10):621–9.

Hubley-Kozey CL, Hatfield GL, Davidson KC. Temporal coactivation of abdominal muscles during dynamic stability exercises. J Strength Condit Res/Natl Strength Condit Assoc 2010;24(5):1246–55.

Jonsson B. Electromyography of the erector spinae muscle. Med Sport: Biomech 1973;III:8294–300.

Kasai Y, Morishita K, Kawakita E, Kondo T, Uchida A. A new evaluation method for lumbar spinal instability: passive lumbar extension test. Phys Ther 2006;86(12):1661–7.
Kavcic N, Grenier S, McGill SM. Determining the stabilizing role of individual torso muscles during rehabilitation exercises. Spine 2004;29(11):1254–65.
Kendall FP, McCreary EK. Muscles, testing and function; 1983.
Lewek MD, Rudolph KS, Snyder-Mackler L. Quadriceps femoris muscle weakness and activation failure in patients with symptomatic knee osteoarthritis. J Orthop Res 2004;22(1):110–5.
MacDonald D, Moseley GL, Hodges PW. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. Pain 2009;142(3):183–8.
Marras WS, Davis KG. A non-MVC EMG normalization technique for the trunk musculature: Part 1. Method development. J Electromyogr Kinesiol 2001;11(1):1–9.
McGill SM. Low back disorders: evidence-based prevention and, rehabilitation; 2002.
Mens JM, Vleeming A, Snijders CJ, Koes BW, Stam HJ. Reliability and validity of the active straight leg raise test in posterior pelvic pain since pregnancy. Spine 2001;26(10):1167–71.
Mirka G, Kelaher D, Baker A, Harrison A, Davis J. Selective activation of the external oblique musculature during axial torque production. Clin Biomech 1997;12(3):172–80.
Nelson-Wong E, Callaghan JP. Is muscle co-activation a predisposing factor for low back pain development during standing? A multifactorial approach for early identification of at-risk individuals. J Electromyogr Kinesiol 2010;20(2):256–63.
Nie H, Arendt-Nielsen L, Andersen H, Graven-Nielsen T. Temporal summation of pain evoked by mechanical stimulation in deep and superficial tissue. Journal Pain 2005;6(6):348–55.
Ostgaard HC, Zetherstrom G, Roos-Hansson E. The posterior pelvic pain provocation test in pregnant women. Eur Spine J 1994;3(5):258–60.
Panjabi MM. A hypothesis of chronic back pain: ligament subfailure injuries lead to muscle control dysfunction. Eur Spine J 2006;15(5):668–76.
Reme SE, Hagen EM, Eriksen HR. Expectations, perceptions, and physiotherapy predict prolonged sick leave in subacute low back pain. BMC musculoskeletal disorders; 2009. p. 10139.
Roland M, Morris R. A study of the natural history of back pain. Part I: development of a reliable and sensitive measure of disability in low-back pain. Spine 1983;8(2):141–4.
Silfies SP, Squillante D, Maurer P, Westcott S, Karduna AR. Trunk muscle recruitment patterns in specific chronic low back pain populations. Clin Biomech 2005;20(5):465–73.
Silfies SP, Bhattacharya A, Biely S, Smith SS, Giszter S. Trunk control during standing reach: a dynamical system analysis of movement strategies in patients with mechanical low back pain. Gait Posture 2009;29(3):370–6.
Spitzer WO, LeBlanc F, Dupuis M. A scientific approach to the assessment and management of activity-related spinal disorders: a monograph for clinicians. Spine 1987;12(Suppl. 75):S3–S59.
Stanton TR, Latimer J, Maher CG, Hancock M. Definitions of recurrence of an episode of low back pain: a systematic review. Spine 2009;34(9):E316–322.
Stuge B, Laerum E, Kirkesola G, Vollestad N. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a randomized controlled trial. Spine 2004;29(4):351–9.
Sullivan MJ, Bishop S, Pivik J. The pain catastrophizing scale: development and validation. Psychol Assess 1995;7(4):524–32.
Sullivan MJ, Thibault P, Savard A, Catchlove R, Kozey J, Stanish WD. The influence of communication goals and physical demands on different dimensions of pain behavior. Pain 2006;125(3):270–7.
Tucker KJ, Hodges PW. Motoneurone recruitment is altered with pain induced in non-muscular tissue. Pain 2009;141(1–2):151–5.
van der Hulst M, Vollenbroek-Hutten MM, Rietman JS, Hermens HJ. Lumbar and abdominal muscle activity during walking in subjects with chronic low back pain: support of the "guarding" hypothesis? J Electromyogr Kinesiol 2010;20(1):31–8.
van Dieen JH, Cholewicki J, Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine 2003;28(8):834–41.
Vezina MJ, Hubley-Kozey CL. Muscle activation in therapeutic exercises to improve trunk stability. Arch Phys Med Rehabil 2000;81(10):1370–9.
Vink P, van der Velde A, Verbout AJ. A functional subdivision of the lumbar extensor musculature: Recruitment patterns and force – EMG relationships under isometric conditions. Electromyogr Clin Neurophysiol 1988:28517–25.
Vleeming A, de Vries HJ, Mens JM, van Wingerden JP. Possible role of the long dorsal sacroiliac ligament in women with peripartum pelvic pain. Acta Obstet Gynecol Scand 2002;81(5):430–6.
Waddell G, Somerville D, Henderson I, Newton M. Objective clinical evaluation of physical impairment in chronic low back pain. Spine 1992;17(6):617–28.
Wasiak R, Kim J, Pransky G. Work disability and costs caused by recurrence of low back pain: longer and more costly than in first episodes. Spine 2006;31(2):219–25.
Wewers ME, Lowe NK. A critical review of visual analogue scales in the measurement of clinical phenomena. Res Nurs Health 1990;13(4):227–36.

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